Comparative Medicine
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Vol 65, No 2
April 2015
Pages 133–139

*Case Report*

# Hepatic Lipidosis in a Research Colony of Big Brown Bats (*Eptesicus fuscus*)

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**During a nearby construction project, a sudden decrease in food intake and guano production occurred in an outdoor colony of big brown bats (*Eptesicus fuscus*), and one animal was found dead. Investigation revealed that the project was generating a large amount of noise and vibration, which disturbed the bats' feeding. Consequently the bats were moved into an indoor enclosure away from the construction noises, and the colony resumed eating. Over the next 3 wk, additional animals presented with clinical signs of lethargy, weight loss, ecchymoses, and icterus and were necropsied. Gross necropsy of the affected bats revealed large, pale yellow to tan, friable livers with rounded edges that floated when placed in 10% neutral-buffered formalin. Some bats had ecchymoses on the webbing and skin and gross perirenal hemorrhage. Histologic examination showed hepatic and renal tubular lipidosis. The clinical and pathologic signs of hemorrhage and icterus were suggestive of hepatic failure. Hepatic lipidosis was attributed to stress and inappetence associated with environmental perturbations. Once the environmental stressor was removed, the colony morbidity and mortality decreased. However, 2 y later, a series of new environmental stressors triggered additional deaths associated with hepatic lipidosis. Over a 9-y period, 21 cases of hepatic lipidosis were diagnosed in this bat colony.**

The big brown bat (*Eptesicus fuscus*), a member of the family Vespertilionidae, is an insectivorous species that relies on echolocation for the identification of prey and navigation during flight.[1,2,19] Several subspecies with specific geographic distributions throughout the continental United States have been identified.[6,24] Brown bats reach a typical adult weight of 13 to 25 g, give birth to 1 or 2 young per year, and hibernate in buildings, crevices, caves, and mines.[19] Uses of this species in biomedical research include studies of spatial memory and flight dynamics, the integration of auditory stimuli through echolocation, and the development of echolocation and communication signals.[1,21,23]

Spontaneously occurring medical problems in big brown bats include predation, trauma, emaciation, and infectious disease.[3] Specific pathogens described in this species in the wild include white-nose syndrome (attributed to infection with *Pseudogymnoascus destructans*), *Pasteurella multocida* serotype 1, *Demodex* mites, rabies virus (*Lyssavirus*), and group 1 (Rocky Mountain bat) coronavirus.[3,6,9,11,15,18] In addition, sporadic cases of cystitis with chronic interstitial nephritis and of hepatic lipidosis have been reported, although the etiology of hepatic lipidosis and concurrent clinical and pathologic findings were not discussed in these reports.[2,18] Three captive horseshoe bats (*Rhinolophus ferrumequinum*) with hepatic, renal, and cardiac lipidosis, presumably related to stress and fat mobilization with excessive deposition of fatty acids in the liver and other organs, have been described.[13]

We here describe a colony of big brown bats that developed lethargy, weight loss, and hemorrhage and were diagnosed with hepatic lipidosis due to exposure to an environmental stressor. The colony morbidity and mortality decreased after intervention; however, 2 y later, a series of new environmental stressors triggered additional deaths associated with hepatic lipidosis. Subsequent colony mortality was tracked, with infrequent cases of hepatic lipidosis occurring over the next 5 y.

*Received: 12 Jul 2014. Revision requested: 18 Sep 2014. Accepted: 03 Dec 2014.*
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## Materials and Methods

The affected colony consists of wild-caught big brown bats (*E. fuscus*) from North Carolina and their offspring, which have been raised in captivity. The colony population varies but comprises 150 to 275 bats in a given year. The bats are used in studies of echolocation and vocalization, and all procedures are approved by the University of Washington IACUC and performed in accordance with the Animal Welfare Act. The research uses anatomic identification and tracing of the different functional pathways involved in the analysis of sound and physiologic and pharmacologic methods to examine the interactions of the multiple parallel pathways at the level of single target neurons. Animals were captured, transported, and maintained in accordance with the Animal Welfare Act. New bats were introduced to the colony in 1999, 2000, and 2005. The bats are group-housed in cloth-draped dens for roosting and are fed daily with live mealworms that are fed a diet of cream of wheat (1 box), dry-milk powder (1 cup), 1 piece of sliced fresh fruit and vegetable (apples, bananas, broccoli, yams, carrots, cauliflower), and Spiru-tein protein mix (1/4 cup; Nature's Plus, Natural Organics, Melville, NY). Mealworms are provided in shallow metal dishes placed on shelves away from the roosting dens. Bats typically are not noted to echolocate to find their food but are observed to echolocate at other times when flying around the enclosure.

The colony is subject to variations in local environmental light and temperature conditions but is shaded and maintained above a minimum temperature of 40 °F by using a heater. The metal and concrete shed enclosure, located on the roof of the animal facility, allows natural light through clear corrugated plastic roof panels. The outer metal walls roll open to allow ventilation and temperature control through an interior mesh. During the winter months, the bats enter torpor with a concurrent decrease in food consumption. The quantity of mealworms provided decreases as consumption decreases, although enough mealworms are provided so that the food supply is not completely empty at any given time. Previous health problems in the colony have included ectoparasites and seasonal infectious and noninfectious distal limb arthropathy (joint swelling). The colony breeds successfully, and, after mating in the fall, approximately 50% of the female bats give birth in the spring. Rabies testing is performed at least annually, on bats that die without an identified cause of death on necropsy. Rabies testing is performed more frequently after newly caught bats are introduced, and the frequency of testing is decreased as the time in captivity increases.

A search of colony mortality reports, medical records, and necropsy records from August 2004 to December 2013 identified 229 cases of deceased adult and juvenile bats, of which necropsy results were available for 45. Necropsies were not performed on all deceased bats, and histology was not performed on all bats that underwent necropsy. Juvenile bats were defined as being younger than 21 d of age or when specified in the medical record as such; juvenile bats accounted for 56 of the cases. Sex, age, and body weight were not available for all bats but were recorded when known or measured. A database of monthly colony mortality records, which included the total population size and the number of bats that died during the previous month, was available from January 2006 to December 2013. The number of bats that died during March 2009 was unavailable; and for this month, the average colony mortality for March was used in statistical analyses. Analyses performed included the variation in population size by month and by year, calculation of the percentage colony mortality by year, and calculation of the percentage colony mortality by month. All statistical analyses were performed by using Prism 5 (GraphPad Software, San Diego, CA). When data were normally distributed, statistical analyses were performed by using one-way ANOVA with the Tukey multiple-comparisons posttest. When data were not normally distributed, statistical analyses were performed by using the Kuskal–Wallis test with the Dunn multiple-comparisons posttest. A $P$ value of less than 0.05 was considered statistically significant.

## Case Report

On August 3, 2004, an animal caretaker found a single dead bat in the outdoor enclosure. Colony-wide reduced food intake and decreased production of guano was noted at that same time. Investigation by veterinary and laboratory personnel found that construction on the building roof where the bats were housed had begun 2 d earlier, resulting in considerable noise at night. To address the concern that the environmental perturbations were the cause of reduced food consumption, the colony was moved that day to a new, noise-reduced holding area inside the building (Table 1). Immediately after the move, the bats resumed eating and producing guano at normal levels. However, the new cages were smaller than the primary housing enclosure, had fewer natural temperature fluctuations during the day, and were inside the building where the construction continued. Therefore, the bats remained potentially subject to increased noise and vibrations. To mitigate these problems, construction was initiated on a large outdoor enclosure in another building, the windows were opened to allow for natural temperature variation, and the interior noise level was monitored.

After the colony had been housed for 3 wk in the indoor facility, the caretakers found 7 dead and a single moribund bat over a 5 d period. Two of the recently deceased animals and the moribund bat were euthanized and necropsied; necropsy revealed severe, diffuse hepatic lipidosis in all 3 animals. Subsequent investigation revealed that the light cycle timer had malfunctioned and that the bats were exposed to continuous light, possibly since entering the new housing area 3 wk earlier. The light-cycle timer was corrected, and the colony was started on nutritional supplementation (KoolAid Jell-O, Kraft Foods, Northfield, IL). Unthrifty bats were isolated from the colony and handfed mealworms daily. Isolated bats were released back into the colony once they had regained adequate body condition.

Appetites increased after initiation of supplementation and handfeeding, but because of difficulty in permanently correcting the light cycle, the bats were moved again (September 2004) to a new indoor room in a different building. During the next few weeks, 6 additional bats were found dead in the cage; the 4 sent to necropsy were found to have hepatic lipidosis. The bats were moved to a temporary outdoor enclosure in a different building in November 2004, and the colony appeared to be stable. During this time period, 5 deaths occurred; the 3 bats necropsied showed no evidence of hepatic lipidosis. Once construction ceased at the primary building, the bats returned to their original outdoor enclosure (February 2005). During this move, bats showing signs of malnutrition (emaciation, dehydration, or lethargy) were individually housed and handfed. After this move, 6 animals died; gross examination of 3 bats was again consistent with hepatic lipidosis. Of the 26 bats that died between August 2004 and March 2005, 13 were 1 y of age or younger and the remaining 13 were older than 1 y; 16 female, 9 male bats were affected, and the sex was not recorded for the remaining bat. Among the 10 bats with hepatic lipidosis, 7 were female, 2 were male; sex was not recorded for 1 bat. Over the next 3 mo, no additional mortality was noted.

## Additional Cases of Hepatic Lipidosis, 2006–2013

A second episode of colony deaths resulting from hepatic lipidosis occurred in late April to early May 2007, when 9 pregnant or postpartum female bats died after changes to reconfigure the colony and during construction on the roof of the building. Previously, the bats had been housed in separate enclosures as 2 colonies according to when the original bats were introduced from the wild; 1 mo prior to the increased morbidity and mortality, the colonies were combined and separated by sex (Table 1, Figure 1). Hepatic lipidosis was diagnosed in all 5 pregnant bats that were sent to necropsy. In addition, 21 juvenile bats died during this same time period. In September 2007, 10 additional deaths occurred, after a census count in late August that required handling of each animal. Hepatic lipidosis was diagnosed after the gross examination of a bat that was submitted to necropsy at this time.

**Table 1.** Mortality and necropsy results in a colony of big brown bats, 2004–2013

| | No. of dead bats | | Necropsy | | |
|---|---|---|---|---|---|
| Year | Adult | Juvenile | No. with hepatic lipidosis / total no. necropsied | Other findings | Events |
| 2004 | 17 | 0 | 7/12 | Thin | 1. Construction near outdoor enclosure (August).<br>2. Move to indoor enclosure (August).<br>3. Light timer malfunction and move to different indoor enclosure (September).<br>4. Move to temporary outdoor enclosure (November). |
| 2005 | 22 | 0 | 3/3 | | 1. Move back to original outdoor enclosure (February).<br>2. 11 mortalities in shipment of 90 wild-caught bats from North Carolina; no necropsies performed (August). |
| 2006 | 9 | 15 | 1/5 | Bronchopneumonia; ventricular hypertrophy; renal mineralization; hepatic fibrosis and lymphocytic histiocytic hepatitis | 1. Hand-reared 2- to 7-d-old pups died due to bloating and dehydration; no additional deaths after changing brand of baby bat food (May). |
| 2007 | 24 | 21 (21 neonates) | 6/8 | Trauma (conspecific); uterine prolapse and endometritis | 1. Construction near outdoor enclosure (April–May)<br>2. Handling of bats for census and separation by sex (September) |
| 2008 | 40 | 0 | 0/2 | Thin; gastritis | 1. National shortage of mealworms (June 2008–January 2009). |
| 2009 | 15 | 0 | 0/1 | Fungal pneumonia; thin | |
| 2010 | 6 | 5 | 0/0 | | |
| 2011 | 17 | 8 (7 neonates) | 2/8 | Pulmonary abscess; joint swell; wing myositis; glossitis; dermatitis; osteomyelitis, bacterial pneumonia | |
| 2012 | 7 | 4 | 0/1 | | |
| 2013 | 16 | 3 | 2/5 | Dystocia; mastitis/enteritis | |
| Total | 173 | 56 | 21/45 | | |

Colony mortality was tracked from 2006 through 2013 (Table 1). During this time, 11 bats were diagnosed with hepatic lipidosis, including the 6 cases previously described (May 2007 and September 2007). An isolated case of hepatic lipidosis was diagnosed in September 2006 and affected an experimental animal that had a cranial implant. In 2008, a national mealworm shortage required a diet change to an insectivorous pellet diet with supplementation of other insects and larvae; 26 bats died after this change (Table 1, Figure 1). The 2 bats sent to necropsy were thin on gross examination, with reduced subcutaneous and intraabdominal body fat stores; however, gross and histopathologic evaluation of the liver was not consistent with hepatic lipidosis in these cases. In 2011, 2 isolated deaths due to hepatic lipidosis were noted. One case involved a female bat with hepatic lipidosis; lesions of the genitalia apparent at necropsy suggested conspecific-induced trauma, which may have led to stress and possible anorexia in the necropsied bat. The other death, in a male bat with hepatic lipidosis, occurred after a census that involved handling of all animals. In 2013, 2 isolated deaths due to hepatic lipidosis were noted and occurred in a young adult male and a juvenile male without a history of known stressors.

From 2006 to 2013, the colony mortality was analyzed by month. For January, February, April, October, November, and December, the mean colony mortality was less than 1%. Specifically,

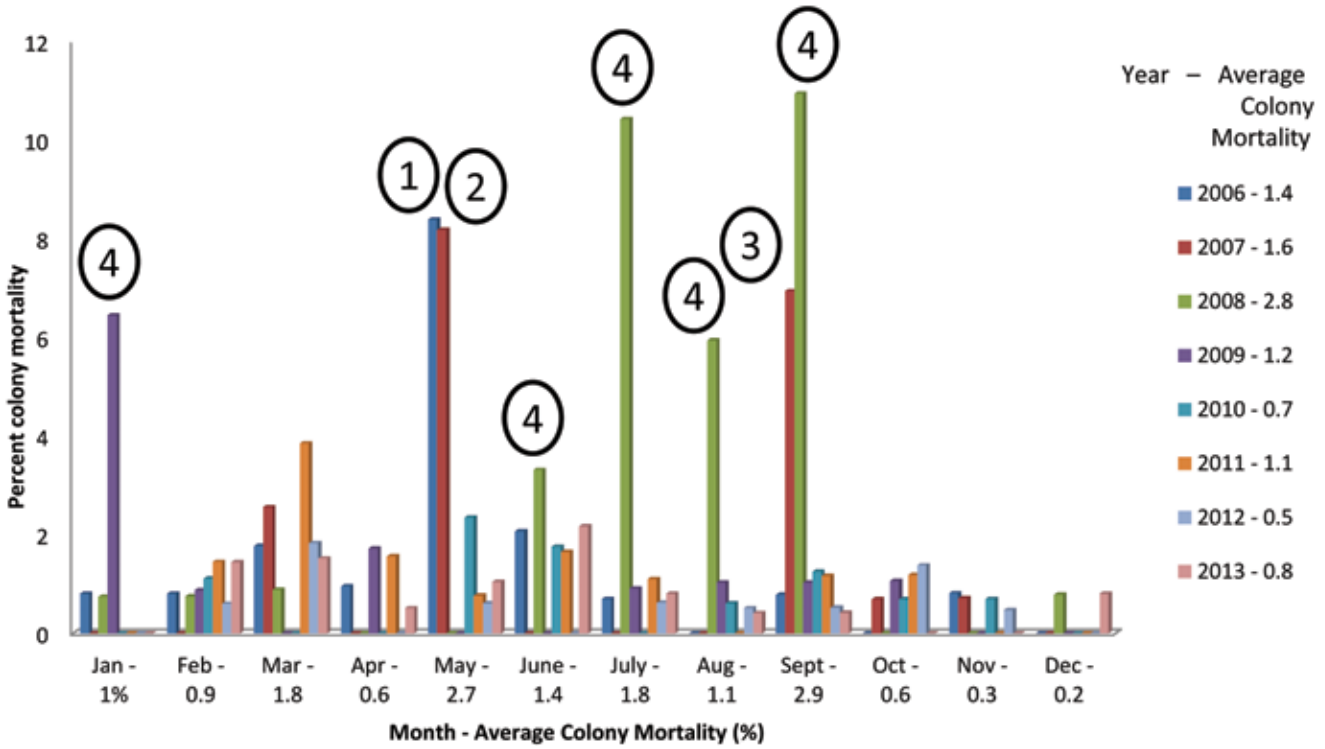


**Figure 1.** Key morbidity and mortality events in a research colony of big brown bats during a 9-y period. (1) Hand-reared 2- to 7-d-old pups died due to bloating and dehydration; (2) 9 pregnant and postpartum bats died during construction near outdoor enclosure; (3) 10 bats died after being handled for census and separation by sex; (4) 40 bats died during national shortage of mealworms.

the months of November and December had the lowest mortalities, with a mean colony mortality of less than 0.4% for these months (Figure 1). Mean colony mortalities were highest for the months of May (2.67%) and September (2.88%; Figure 1) and varied significantly by month ($P = 0.02$) but not by year ($P = 0.70$). The mean colony population varied significantly by year ($P < 0.0001$) and was significantly higher in 2012 and 2013 than in 2006, 2007, 2008, 2009, and 2010. The mean colony population did not vary significantly by month ($P = 0.88$). Sex was recorded for 104 of the 229 bats that died between August 2004 and December 2013; 43 were male, and 61 were female.

## Pathologic Findings

Between August 2004 and August 2013, 45 bats were submitted for postmortem examination, of which 21 bats were diagnosed with diffuse, severe hepatic lipidosis. Of these bats, 14 were female, 4 were male, and sex was not recorded for 3 bats. Body weights were available for 7 nonpregnant adult bats diagnosed with hepatic lipidosis, and the body weight (mean ± 1 SD) was 18.2 ± 3 g (range, 13.5 to 22.0 g). Body condition was noted as poor, with decreased internal and subcutaneous fat deposits, for 4 of the 5 pregnant female bats diagnosed with hepatic lipidosis. Among nonpregnant female and male bats with a necropsy diagnosis of hepatic lipidosis, body condition score was noted as thin in 3 and as good (body condition score of 2.5 on a scale of 5) in 4 bats. Externally, some bats were icteric and had regionally extensive petechiation and ecchymoses on the webbing and skin (Figure 2 A), with gross perirenal hemorrhage (data not shown).

In bats with hepatic lipidosis, the livers were large, pale yellow to tan, and friable with rounded edges (Figure 2 B). Sections of liver floated when placed in 10% neutral buffered formalin. Hepatomegaly was marked in several animals, with the liver comprising as much as 10% of the total body weight. Animals had little or no feces in the intestine and lacked stomach contents. One bat also had gross hemorrhage around the submandibular salivary glands. Histology demonstrated severe, diffuse cytoplasmic distention of hepatocytes by large, round, clear vacuoles that displaced the nucleus (Figure 2 C and D); these vacuoles were negative for periodic acid–Schiff staining, consistent with lipid. There was mild to moderate cholestasis.

The kidney contained moderate, multifocal large, round, clear vacuoles and abundant eosinophilic acellular homogenous proteinaceous material that expanded Bowman's space and moderate, multifocal to coalescing, cytoplasmic distention of cortical renal tubular epithelial cells by large, round, clear vacuoles similar to those in the liver (Figure 2 E and F). In addition, minimal, multifocal, intracytoplasmic lipid vacuoles in cardiomyocytes were noted in 2 animals (data not shown).

Additional abnormalities identified on necropsy in the 24 bats that did not have hepatic lipidosis included fight wounds, arthropathy, mastitis, enteritis, uterine prolapse, and pneumonia (Table 1).

## Discussion

Here we describe 21 cases of mortality that occurred over a 9-y period in a research colony of *E. fuscus* in which hepatic lipidosis

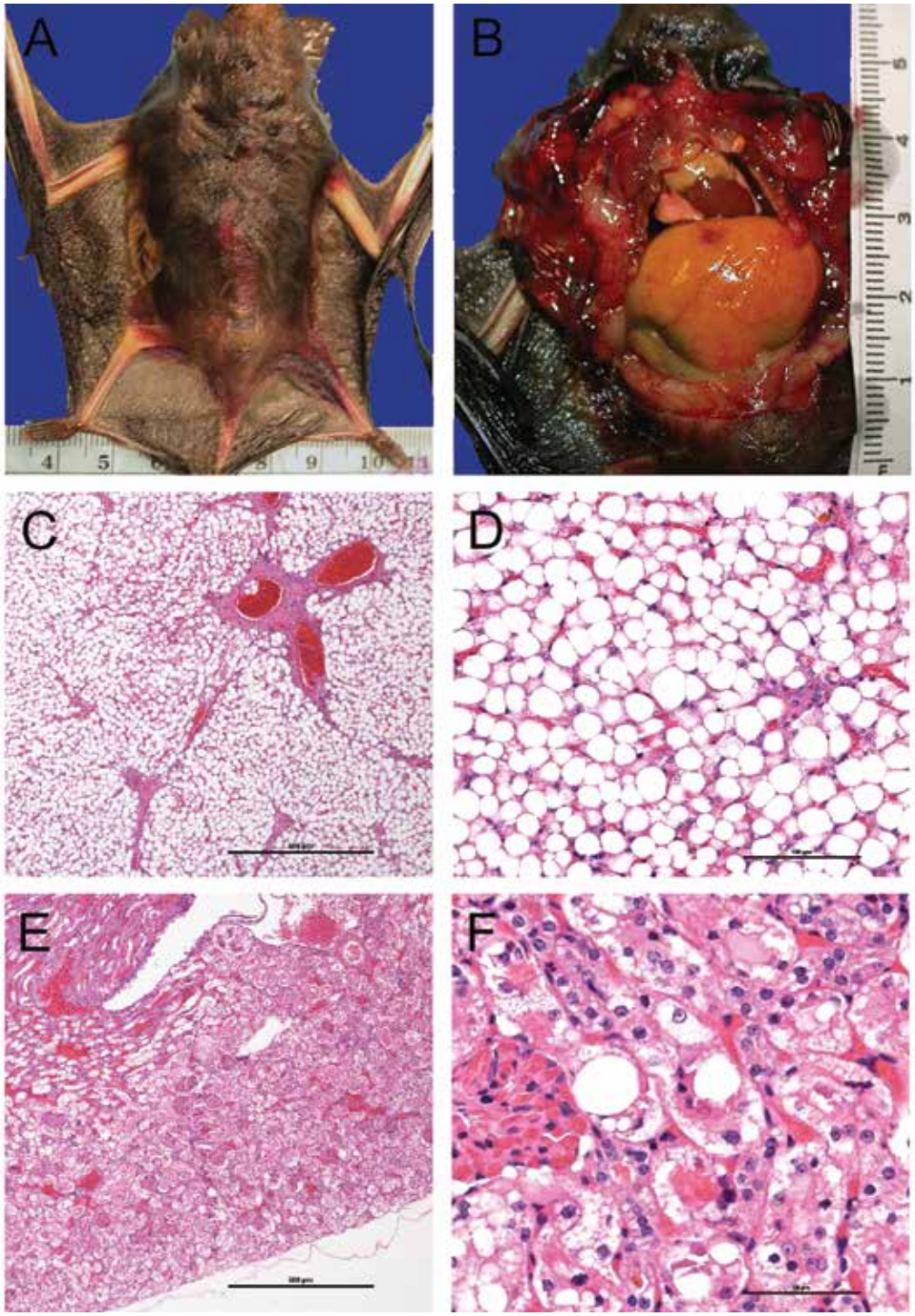


**Figure 2.** (A) Gross appearance of a big brown bat with regionally extensive petechiation and ecchymoses on the webbing and skin of the ventral abdomen. (B) Hepatic lipidosis in the same bat as in Figure 2. The liver is markedly and diffusely enlarged with a yellow, greasy appearance. (C) Histologic appearance of hepatic lipidosis. There is severe, diffuse cytoplasmic distention of hepatocytes by large, round, clear vacuoles that displace the nucleus. Bar, 500 µm. (D) Hepatic lipidosis; higher magnification image of panel C. Bar, 100 µm. (E) Histologic appearance of renal lipidosis in a big brown bat found dead in the colony. Mild autolytic change is present. Bar, 500 µm. (F) Renal lipidosis in a big brown bat; higher magnification image of panel E, showing the multifocal large round clear vacuoles in the glomeruli and tubules with eosinophilic acellular homogenous material in the tubules (protein). Mild autolytic change is present. Bar, 50 µm.

was confirmed on postmortem examination. The majority (19 of 21) of these cases occurred after exposure to an environmental stressor, such as nearby construction or capture. However, hepatic lipidosis also was associated with other systemic illness or conspecific trauma. The pathologic findings included variably severe emaciation and marked, diffuse hepatic lipidosis. In many cases, icterus and hemorrhage were present as well.

The pathogenesis of hepatic lipidosis is complex and multifactorial.[26,28] In veterinary medicine, clinically significant classic hepatic lipidosis in cats, macaques, and postparturient bovines and usually occurs in an overconditioned animal after anorexia, with mobilization of fatty acids by the liver and hepatocyte triglyceride accumulation. [4,20,25,28]Lipidosis occurs when the rate of triglyceride accumulation in the liver overwhelms the triglyceride degradation capacity.[25,28]With subsequent hepatic failure, hepatocyte degeneration results in decreased production of proteins, including clotting factors, which can lead to hemorrhage.[7] Cholestasis caused by hepatocyte distention leads to hyperbilirubinemia and clinical icterus,[20,28] as occurred in many of the affected bats in the current report.

Hepatic lipidosis can occur subsequent to metabolic derangement and endocrine diseases such as diabetes mellitus and hypothyroidism and after exposure to toxins such as aflatoxin, amanita, and carbon tetrachloride.[28] Fat deposition in the liver can be a normal physiologic state in some species, including reptiles.[8] In the majority of the bats described in the current report, hepatic lipidosis developed after exposure to an environmental stress or to illness resulting in anorexia. Necropsy revealed no evidence of primary hepatic, gastrointestinal, renal disease, or endocrinopathy that might have predisposed these animals to disease, and there was no known history of exposure to toxins associated with the construction or with the enclosure itself. Interestingly, large colonies of song sparrows (*Melospiza melodia*) were housed on the roof of the same building as the bats and therefore were subject to the same potential environmental stressors, but no significant morbidity or mortality in the bird colonies occurred during this time. Construction took place in the evening, when the bats feed normally, and the timing of this work likely explains why the bats were affected but not the birds, which are diurnal feeders. In addition, the construction noise may have included intense high-frequency components, to which the bats are highly sensitive.

Similar to this case study, hepatic lipidosis (with concurrent renal and cardiac lipidosis) in 3 captive greater horseshoe bats (*Rhinolophus ferrumequinum*) occurred 5 to 6 mo after capture, and the etiology was presumed to be due to stress and mobilization of fat stores.[13] In big brown bats (*E. fuscus*), hepatic lipidosis has previously been reported only in wild-caught animals, and the etiology was not examined.[2,18] However, in one report, hepatic lipidosis was identified in 9 of 14 *E. fuscus* bats necropsied, indicating that this pathologic finding may not be an unusual finding in this species.[18] Perhaps big brown bats react differently to stress-induced anorexia compared with fasting due to food deprivation (in the absence of environmental stress). Indeed, a national mealworm shortage in 2008 in our colony resulted in several deaths in bats that did not adapt well to an alternative diet, but none of those bats had changes consistent with hepatic lipidosis on postmortem examination. Furthermore, the bats that died during the mealworm shortage were not overconditioned prior to energy restriction and were therefore less predisposed to develop hepatic lipidosis, as has been proposed to occur in cats;[25] alternatively hepatic lipidosis may not have been detected due to the small sampling size, in that only 2 bats were sent to necropsy.

In the wild, insectivorous bats eat a variety of insects which differ in their nutritional content.[27] Previous studies performed in different geographic locations have shown that beetles comprise approximately 60% of the natural diet of *E. fuscus*, with scarab beetles predominating in some studies and juveniles feeding on a wider variety of prey items.[14,27] In captivity, insectivorous bats are often fed a diet of fortified mealworms, the larval form of *Tenebrio molitor*.[2,13] A study on the nutritional characteristics of unfortified mealworms showed them to be an adequate source of most minerals and essential amino acids, although they were deficient in calcium, taurine, vitamin A, vitamin D3, vitamin E, and vitamin B12.[10] Fortified mealworms have an approximate fat content of 27%, which most likely is higher than that of the more varied insect diet consumed in the wild and may predispose bats to obesity.[2,5,10,13] Conversely, captive bats may be underweight compared with wild bats because captive bats can be less inclined to consume prey such as mealworms that do not stimulate echolocation and normal foraging behavior.[5] Whether the fortified mealworm diet contributed to the episodes of hepatic lipidosis in the bats we describe in the current report is unclear, although there may be other nutritional benefits to eating a variety of insect species compared with the consumption of a single-species fortified invertebrate food source.

The current study revealed a temporal trend in the colony, with the highest mortality and incidence of hepatic lipidosis during the late spring and late summer months. Two episodes of illness, in September 2004 and September 2007, occurred at times of the year when the bats normally gain weight and deposit fat in anticipation of torpor. In another insectivorous bat species, *Scotophilus heathi*, increased corticosterone levels during September and October cause hyperinsulinemia and increased fat deposition from in October and November.[22] In addition, the *Scotophilus* bats show increasing triglyceride, free fatty acid, and serum cholesterol levels from July to December,[22] possibly predisposing animals to pathologic lipidosis when an environmental stressor occurs and food intake decreases. Mortality in our bat colony was significantly increased during the month of May also, and in one episode of mortality during this month, pregnant bats were affected exclusively. Ketosis in pregnant and lactating animals is well described in many veterinary species and occurs when free fatty acids are released from body fat stores and esterified into fatty acyl CoA in the liver during times of stress in female animals with increased metabolic demands.[28] Compared with other pregnant animals, big brown bats may be even more sensitive to calorie restriction, given their high frequency of twinning, large fetal size relative to maternal weight, and the caloric demands required for flight.[17] In addition, female insectivorous bats rely more on carbohydrate metabolism than do their male counterparts and are more efficient at storing energy, which may render them more susceptible to hepatic lipidosis during times of high metabolic stress, such as gestation and lactation.[12,22]

We hypothesize that big brown bats respond to stress-induced anorexia by liberating peripheral fat lipid stores, which are absorbed by the liver. Hepatic lipidosis is associated with a fatal-fasting-like syndrome in other mammalian species, including monkeys and cats,[4,28] and *E. fuscus* appears to respond in a similar manner. During the episodes of high mortality and nutritional stress, this colony was treated by providing simple nutritional

supplements, isolation and handfeeding of ill animals, and removal or avoidance of environmental stressors. Specific therapy such as carnitine or S-adenosyl methionine was not used in the bats in this report, although these supplements seem to decrease the incidence of hepatic lipid accumulation in cats on dietary restriction studies.[25] To our knowledge, the use of these supplements in vespertilionid bat species has not been described. In the absence of major environmental perturbations, the baseline mortality associated with hepatic lipidosis was low, although sporadic cases were noted. As evidenced by the findings from a large number of necropsy-confirmed cases over a 9-y period, big brown bats (*E. fuscus*) appear to be particularly susceptible to stress-induced anorexia and the development of hepatic lipidosis.

## Acknowledgments

We acknowledge Douglas Fitts for his advice regarding statistical analysis.

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